

James Penn¹, Zbigniew Zylicz^{1, 2}

¹Hospice in the Welad, Maidstone Road, Pembury, United Kingdom

²Dove House Hospice, Hull, United Kingdom

Shoulder pain in terminally ill? Think of suprascapular nerve entrapment!

Abstract

Many terminally ill patients suffer because of poorly controlled musculoskeletal pain. Oral and parenteral systemic analgesics are usually ineffective in this type of pain. It is therefore important to diagnose and treat accordingly. Patients with shoulder pain may suffer from suprascapular nerve entrapment, a syndrome known from sport and occupational medicine. Within, we describe this syndrome, mechanism of onset, diagnosis and treatment. Simple injection of local anaesthetics and corticosteroids may have an effect for several weeks.

Key words: suprascapular nerve entrapment, musculoskeletal pain, advanced disease, opioid responsiveness

Introduction

Musculoskeletal pain is common in end stages of many chronic diseases. Long periods of immobility, poor nutrition and cachexia are known causes of these complaints. However not many clinicians realize that part of these pain symptoms can be due to nerve entrapment. Factors that contribute to poorly understood musculoskeletal pain are: weak muscles, loss of subcutaneous fat tissue, increased joint mobility and compression against bony edges. This pain is not only difficult to diagnose but also difficult to treat with conventional oral and parenteral (co-)analgesics.

In this article we describe one of the common musculoskeletal pain syndromes which depends on the traction of the suprascapular nerve. After considering its anatomy and physiology we shall explain the mechanism of the onset of pain and using this knowledge, propose treatment.

Functional anatomy

The shoulder blade (scapula) is an important bone in the shoulder girdle. It connects the shoulder with chest and accommodates the shoulder movement. The triangle-shaped shoulder blade is on one side connected by joints with the humerus and collar-bone (clavicula) and on the other side connected by strong muscles with the chest and spine.

At the upper part of the shoulder blade there is an incision, the suprascapular notch, covered by a suprascapular ligament (Fig. 1). This is the place of entrance of the suprascapular nerve which originates from the brachial plexus (C₅, C₆ and sometimes C₄) [1]. This nerve carries motor fibres for the supra- and infra-scapular muscles, but sends sensory branches to both the glenohumeral and acromioclavicular joint. It does not innervate the skin. The suprascapular nerve divides and part of it enters the infrascapular fossa and goes to the shoulder joint.

Address for correspondence: Zbigniew Zylicz
Dove House Hospice, Hull, HU8 8DH, United Kingdom
e-mail: b.zylicz@dovehouse.org.uk



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Figure 1. Suprascapular nerve originates in brachial plexus

The anatomy of the suprascapular notch has individual variance, from wide and “open” to narrow and hole-like [2]. It is here where the nerve can become entrapped and inflamed producing irritation and pain. The other place when similar reaction can take place is the narrow space between the supra- and infra-scapular fossa. This second situation is of less importance to palliative care and we shall leave it out of the scope of this article.

Mechanism of suprascapular nerve entrapment (SSNE)

Shoulder movement can increase or decrease traction on the suprascapular nerve. The maximal traction is generated when you ask the patient to reach, with his arm (his elbow at the level of his eyes) to the contralateral scapula (Fig. 2). This is called Thompson and Kopell test. The patient with SSNE will experience pain in his shoulder.

Repeated traction of the nerve can occur during overhead training (e.g. baseball, volleyball, basketball or athletics) [3, 4]. Accidental lesions like scapula fracture are known reasons of suprascapular nerve dysfunction and neuropathy [5]. Repeated movements with shoulders (assembly lines) put the workers at risk of SSNE.

In palliative care there is a different mechanism probably more important [6]. Due to weak rotatory cuff muscles, winging of the scapula increases causing repeated traction of the suprascapular nerve. This is especially the case when the patient overloads their shoulders. See Table 1.

Tumour growth, both malignant and benign, that directly compresses the suprascapular nerve is seldom encountered [7, 8].

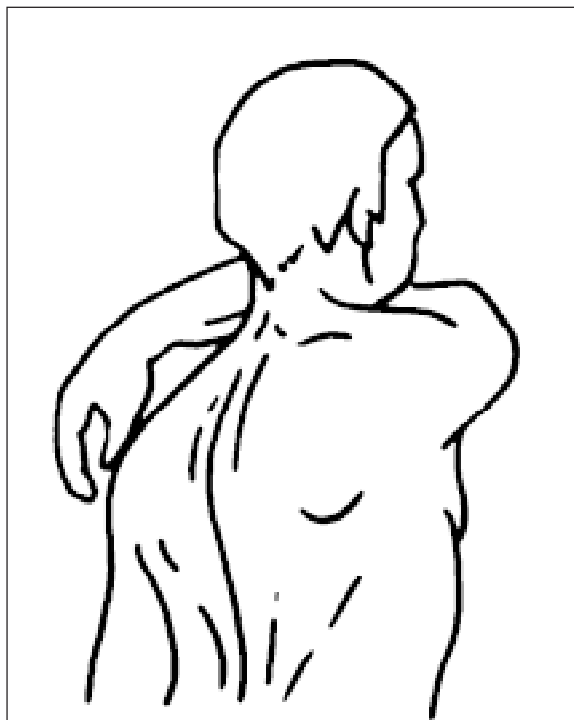


Figure 2. Thompson and Kopell test. Ask patient to reach with his arm to the contralateral shoulderblade

Table 1. Situations where SSNE can be expected and/or anticipated in palliative care

Weakness and cachexia
Spinal lesions with paraparesis
Patients using wheelchairs
Patients using crutches
Patients with lymphoedema
Patients lying on one side (with lung, pleura or liver pathology) by default
Dyspnoeic patients using their accessory muscles to ease breathing

How to make the diagnosis?

Patients usually complain of uni-, or occasionally bilateral shoulder pain. The pain is typically resistant to classic analgesia although some drugs may be helpful for some time (see therapy). Patient lying on one side may complain of sleeplessness with nocturnal pain. Overhead movements may be extremely painful and the patient may avoid these. The Thompson and Kopell test is helpful but not discriminative. Pain on pressure with the index finger on the suprascapular fossa will add to the diagnosis. Disproportionate atrophy of the supra- and infraspinatus muscles (uni- or bilaterally) is very suggestive for SSNE [9, 10]. Winging of the scapula (scapula alata) may

also be evident. In some cases, diagnosis is difficult but can be confirmed by resolution following treatment.

Treatment

As previously mentioned, systemic analgesics are often ineffective, especially when the measures to relieve the burden are not undertaken. The nature of the nerve entrapment is usually inflammatory and maximal in the suprascapular notch; anti-inflammatory drugs may help initially, but can mask the pain and delay diagnosis. The most important aspect of treatment is prevention of recurrence. This may involve special equipment like electric wheelchairs, hoists and slings) and teaching patients how to use them.

In some patients, usually not the terminally ill, with specific anatomic variants, surgical treatment may be appropriate. Nerve decompression is effective in most of these patients [11].

Local treatment is by a nerve blockade. This may be performed by a skilled anaesthesiologist using neurodestructive measures, e.g. 10% phenol, but more often patients would be seen at the bedside by the non-anaesthesiologist for infiltration with several millilitres of bupivacaine (0.5%) and corticosteroids, e.g. methylprednisolone [12]. In most patients this treatment is sufficient and the pain may remain controlled for as long as 2–3 months. Injecting the latter drug combination one should think about the bi-phasic effect. At first bupivacaine would act as a local anaesthetic and the pain can be relieved within 20–30 minutes lasting for 12–24 hours. A good response in the first 30 minutes gives indication of correct placement/diagnosis. Depo-corticosteroids may need some time to diffuse to the right place and the onset of the effect may be delayed for 2–3 days. So there may be a gap and after initial good analgesia the pain may reappear and disappear later again. Repeated injections are seldom needed but possible. After two or three injections, every 2–3 months corticosteroids may induce marked atrophy. Thus after second procedure neurodestructive blockade might be more reasonable choice.

Allow the patient to sit straight up. Never do the injection with the patient lying flat in bed. As the Depo-medrone sediments from bupivacaine you should keep the syringe up, in the way the heavier Depo-medrone will be injected

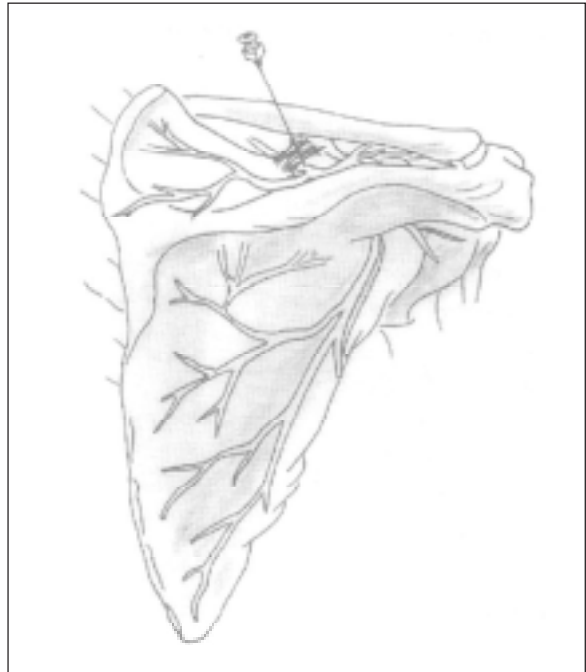


Figure 3. Technique of infiltration of the suprascapular notch with bupivacaine and depo-corticosteroids

first. Do not perform injection in patients with lymphoedema.

The technique is simple (Fig. 3). Mark with your nail the point of maximal pain. Localise with your non dominant hand the bony crista. Try to introduce the needle to touch the Crista fist. Move the needle in the presumable direction of suprascapular notch “walking” with the needle 2–3 mm at a time. Do not allow the needle to go into the canal. The patient may help identifying the moment of increased pain. Always aspirate to check for puncturing of the blood vessel. Inject slowly changing from time to time position of the needle.

The treatment has its own limitations and adverse effects. Do not try to enter with the needle in the suprascapular canal as there is a real chance of damage to the nerve or significant haematoma. Injection with a long needle, deep in the tissues has a potential of pneumothorax. In difficult cases SSN blockade can be performed under CT control. This is sometimes needed with different anatomical variants [13, 14].

Another consideration is the placement of a fine bore catheter in the region of the suprascapular nerve, infusing local anaesthetics. This may be useful in the treatment of an inoperable fracture of the upper humerus [15] in the dying patient.

Conclusion

SSNE may be the reason for some if not many shoulder pains in the terminally ill. However the epidemiology of this phenomenon is still unknown. Lack of awareness may cause underdiagnosis. The symptoms of pain on compression of suprascapular notch, pain on adduction of the arm and unbalanced atrophy of the supraspinatus muscle may help to make the diagnosis. Many risk factors frequently encountered in the terminally ill may be identified. The treatment can be done at the bedside by a skilled doctor using the techniques described. It is not dangerous and only a few minor complications are known. We hope that this article will contribute to increased awareness about this syndrome amongst the professionals in palliative care.

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